WHAT IS CLAIMED IS:

1. A method of treating, ameliorating or preventing a disease or condition caused by exposure to radionuclides, biological agents, or chemical agents in an animal, comprising administering to an animal in need thereof an effective amount of a caspase inhibitor such that cell death in response to said exposure to said radionuclides, biological agents, or chemical agents is inhibited.

2. The method of claim 1, wherein said cell death occurs in cells of the gastrointestinal tract, skin, hair, bone marrow, immune system, nervous system or liver.

3. The method of claim 1, wherein said caspase inhibitor is administered topically or orally.

4. The method of claim 1, wherein said caspase inhibitor is administered systemically by intravenous, intraperitoneal, intramuscular, or subcutaneous injection.

5. The method of claim 1, wherein said caspase inhibitor is administered as part of a pharmaceutical composition comprising a pharmaceutically acceptable carrier.

6. The method of claim 1, wherein said exposure to radionuclides, biological agents, or chemical agents is unintentional.

7. The method of claim 6, wherein said radionuclides, biological agents, or chemical agents are from a nuclear power plant, manufacturing or processing plant, research facility, or hospital.

8. The method of claim 1, wherein said exposure to radionuclides, biological agents, or chemical agents is intentional.

9. The method of claim 8, wherein said radionuclides, biological agents, or chemical agents are from a spill or a bomb.

10. The method of claim 1, wherein said radionuclides are part of a radiopharmaceutical agent.

11. The method of claim 1, wherein said radionuclides are selected from the group consisting of actinium (^{225}Ac), americium (^{241}Am), antimony (^{124}Sb, ^{125}Sb), arsenic (^{72}As, ^{73}As, ^{74}As), astatine (^{211}At), barium (^{103}Ba, ^{140}Ba), beryllium (^{7}Be), bismuth (^{206}Bi, ^{207}Bi, ^{212}Bi, ^{213}Bi), bromine (^{77}Br), cadmium (^{109}Cd, ^{115}Cd), calcium (^{45}Ca), carbon (^{14}C), cerium (^{139}Ce, ^{141}Ce, ^{144}Ce), cesium (^{129}Cs, ^{137}Cs), chromium (^{51}Cr, ^{56}Cr), cobalt (^{55}Co, ^{56}Co, ^{57}Co, ^{58}Co, ^{60}Co, ^{64}Co), copper (^{61}Cu, ^{64}Cu, ^{67}Cu), erbium (^{169}Er), europium (^{152}Eu), fluorine (^{18}F), gadolinium (^{153}Gd), gallium (^{67}Ga, ^{68}Ga), gold (^{195}Au, ^{198}Au, ^{199}Au), hafnium (^{175}Hf, ^{181}Hf), holmium (^{166}Ho), hydrogen (^{3}H), krypton (^{85}Kr), iodine (^{123}I; ^{125}I, ^{126}I, ^{131}I, ^{133}I), indium (^{111}In, ^{113}In), iridium (^{192}Ir), iron (^{52}Fe, ^{55}Fe, ^{59}Fe), lead (^{203}Pb, ^{210}Pb, ^{212}Pb), lutetium (^{177}Lu), magnesium (^{52}Mg), manganese (^{54}Mn), mercury (^{197}Hg, ^{203}Hg), molybdenum (^{99}Mo), neodynium (^{147}Nd), neptunium (^{237}Np), nickel (^{57}Ni, ^{63}Ni), niobium (^{95}Nb), osmium (^{185}Os, ^{191}Os), palladium (^{103}Pd, ^{109}Pd), phosphorus (^{32}P, ^{33}P), platinum (^{195}Pt, ^{197}Pt), plutonium (^{239}Pu), potassium (^{40}K), praseodynium (^{142}Pr, ^{143}Pr), promethium (^{147}Pm), protactinium (^{233}Pa), radium (^{223}Ra, ^{226}Ra), rhenium (^{186}Re, ^{188}Re), rhodium (^{105}Rh), rubidium (^{81}Rb, ^{86}Rb), ruthenium (^{95}Ru, ^{97}Ru, ^{103}Ru, ^{105}Ru, ^{106}Ru), samarium (^{153}Sm), scandium (^{44}Sc, ^{46}Sc, ^{47}Sc), selenium (^{72}Se, ^{73}Se, ^{75}Se), silver (^{100}Ag, ^{111}Ag), sodium (^{22}Na), strontium (^{85}Sr, ^{89}Sr, ^{90}Sr), sulfur (^{35}S), tantalum (^{179}Ta, ^{182}Ta), technetium (^{99}Tc), tellurium (^{121}Te, ^{122}Te, ^{125}Te, ^{132}Te), terbium (^{161}Tb), thalium (^{170}Tl, ^{201}Tl, ^{204}Tl), thorium (^{228}Th, ^{230}Th, ^{232}Th), thulium (^{165}Tm,^{167}Tm, ^{168}Tm,

^{170}Tm), tin (^{113}Sn), titanium (^{44}Ti), tungsten (^{185}W), uranium(^{233}U, ^{235}U, ^{238}U), vanadium (^{48}V, ^{49}V), ytterbium (^{169}Yb), yttrium (^{88}Y,^{90}Y,^{91}Y), zinc (^{62}Zn, ^{65}Zn) and zirconium (^{95}Zr).

12. The method of claim 1, wherein said biological agents are selected from the group consisting of anthrax and its toxins, botulinum and its toxins, aflatoxin, sterigmatocystin, deoxynivalenol, fumonisin B1, *Clostridium dificile* and its toxins, plague (*Yersinia pestis*) and its toxins, hemorrhagic fevers, *Staphylococcus aureus*, Streptococcus, ricin, modeccin, diphtheria, and Pseudomonas, and cholera and its toxins.

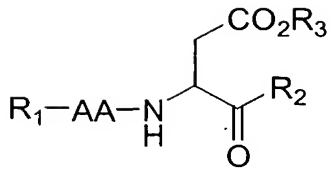
13. The method of claim 1, wherein said chemical agents are selected from the group consisting of phosphoramide mustard, melphalan, chlorambucil, quinacrine mustard, nitrogen mustard, cyclophosphamide, 4-hydroxycyclophosphamide, and cyanide.

14. The method of claim 1, wherein said caspase inhibitor is administered after exposure to radionuclides, biological agents, or chemical agents in said animal.

15. The method of claim 1, wherein said caspase inhibitor is administered during exposure to radionuclides, biological agents, or chemical agents in said animal.

16. The method of claim 1, wherein said caspase inhibitor is administered prior to exposure to radionuclides, biological agents, or chemical agents in said animal.

17. The method of claim 1, wherein said caspase inhibitor has the formula:



(I)

or a pharmaceutically acceptable salt thereof;

wherein R_1 is an N-terminal protecting group;

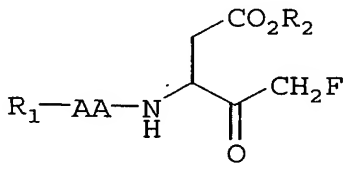
AA is a residue of any natural or non-natural α-amino acid, β-amino acid, derivatives of an α-amino acid or β-amino acid;

R_2 is H or CH_2R_4 where R_4 is an electronegative leaving group; and

R_3 is alkyl or H.

18. The method of claim 17, wherein said caspase inhibitor is Boc-Ala-Asp-CH_2F, Boc-Val-Asp-CH_2F, Boc-Leu-Asp-CH_2F, Ac-Val-Asp-CH_2F, Ac-Ile-Asp-CH_2F, Ac-Met-Asp-CH_2F, Cbz-Val-Asp-CH_2F, Cbz-β-Ala-Asp-CH_2F, Cbz-Leu-Asp-CH_2F, Cbz-Ile-Asp-CH_2F, Boc-Ala-Asp(OMe)-CH_2F, Boc-Val-Asp(OMe)-CH_2F, Boc-Leu-Asp(OMe)-CH_2F, Ac-Val-Asp(OMe)-CH_2F, Ac-Ile-Asp(OMe)-CH_2F, Ac-Met-Asp(OMe)-CH_2F, Cbz-Val-Asp(OMe)-CH_2F, Cbz-β-Ala-Asp(OMe)-CH_2F, Cbz-Leu-Asp(OMe)-CH_2F or Cbz-Ile-Asp(OMe)-CH_2F.

19. The method of claim 1, wherein said caspase inhibitor has the formula II:



(II)

or a pharmaceutically acceptable salt thereof;

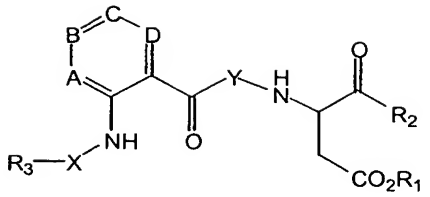
wherein R_1 is an N-terminal protecting group;

AA is a residue of a non-natural α-amino acid or β-amino acid; and

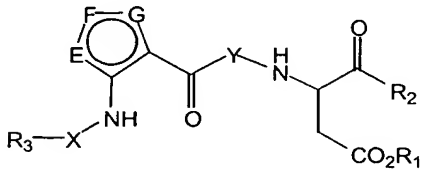
R_2 is an optionally substituted alkyl or H.

20. The method of claim 19, wherein said caspase inhibitor is Boc-Phg-Asp-fmk, Boc-(2-F-Phg)-Asp-fmk, Boc-(F_3-Val)-Asp-fmk, Boc-(3-F-Val)-Asp-fmk, Ac-Phg-Asp-fmk, Ac-(2-F-Phg)-Asp-fmk, Ac-(F_3-Val)-Asp-fmk, Ac-(3-F-Val)-Asp-fmk, Z-Phg-Asp-fmk, Z-(2-F-Phg)-Asp-fmk, Z-(F_3-Val)-Asp-fmk, Z-Chg-Asp-fmk, Z-(2-Fug)-Asp-fmk, Z-(4-F-Phg)-Asp-fmk, Z-(4-Cl-Phg)-Asp-fmk, Z-(3-Thg)-Asp-fmk, Z-(2-Fua)-Asp-fmk, Z-(2-Tha)-Asp-fmk, Z-(3-Fua)-Asp-fmk, Z-(3-Tha)-Asp-fmk, Z-(3-Cl-Ala)-Asp-fmk, Z-(3-F-Ala)-Asp-fmk, Z-(F_3-Ala)-Asp-fmk, Z-(3-F-3-Me-Ala)-Asp-fmk, Z-(3-Cl-3-F-Ala)-Asp-fmk, Z-(2-Me-Val)-Asp-fmk, Z-(2-Me-Ala)-Asp-fmk, Z-(2-*i*-Pr-β-Ala)-Asp-fmk, Z-(3-Ph-β-Ala)-Asp-fmk, Z-(3-CN-Ala)-Asp-fmk, Z-(1-Nal)-Asp-fmk, Z-Cha-Asp-fmk, Z-(3-CF_3-Ala)-Asp-fmk, Z-(4-CF_3-Phg)-Asp-fmk, Z-(3-Me_2N-Ala)-Asp-fmk, Z-(2-Abu)-Asp-fmk, Z-Tle-Asp-fmk, Z-Cpg-Asp-fmk, Z-Cbg-Asp-fmk, Z-Thz-Asp-fmk, Z-(3-F-Val)-Asp-fmk, or Z-(2-Thg)-Asp-fmk.

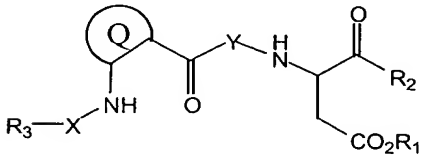
21. The method of claim 1, wherein said caspase inhibitor has the formula of one of III, IV and V:



(III)



(IV)



(V)

or a pharmaceutically acceptable salt thereof;

wherein R_1 is an optionally substituted alkyl or hydrogen,

R_3 is an N-protecting group;

R_2 is hydrogen or optionally substituted alkyl;

A is CR_6 or nitrogen;

B is CR_7 or nitrogen;

C is CR_8 or nitrogen;

D is CR_9 or nitrogen;

provided that not more than two of A, B, C or D is nitrogen; and

R_6-R_9 independently are hydrogen, halo, C_1-C_6 haloalkyl, C_6-C_{10} aryl, C_4-C_7 cycloalkyl, C_1-C_6 alkyl, C_2-C_6 alkenyl, C_2-C_6 alkynyl, C_6-C_{10} aryl(C_1-C_6)alkyl, C_6-C_{10} aryl(C_2-C_6)alkenyl, C_6-C_{10} aryl(C_2-C_6)alkynyl; C_1-C_6 hydroxyalkyl, nitro, amino, cyano, C_1-C_6 acylamino, hydroxy, C_1-C_6 acyloxy, C_1-C_6 alkoxy, alkylthio, or carboxy; or

one of R_6 and R_7, or R_7 and R_8, or R_8 and R_9 are taken together with the carbon atoms to which they are attached to form a carbocycle or heterocycle;

E is CR_{14}, nitrogen, oxygen or sulfur;

F is CR_{15}, nitrogen, oxygen or sulfur;

G is C_{16}, nitrogen, oxygen or sulfur;

provided that only one of E, F, G is nitrogen, oxygen or sulfur, where R_{14}-R_{16} are independently hydrogen, halo, C_1-C_6 haloalkyl, C_6-C_{10} aryl, C_4-C_7 cycloalkyl, C_1-C_6 alkyl, C_2-C_6 alkenyl, C_2-C_6 alkynyl, C_6-C_{10} aryl(C_1-C_6)alkyl, C_6-C_{10} aryl(C_2-C_6)alkenyl, C_6-C_{10} aryl(C_2-C_6)alkynyl; C_1-C_6 hydroxyalkyl, nitro, amino, cyano, C_1-C_6 acylamino, hydroxy, C_1-C_6 acyloxy, C_1-C_6 alkoxy, alkylthio, or carboxy; or

one of R_{14} and R_{15}, or R_{15} and R_{16}, are taken together with the carbon atoms to which they are attached to form a carbocycle or heterocycle;

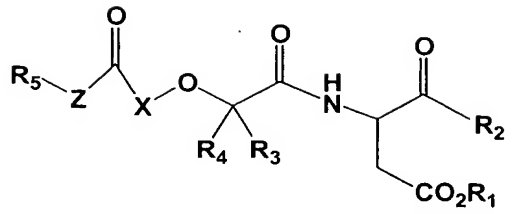
Q represents an optionally substituted saturated or partially saturated carbocycle or heterocycle;

X is a peptide of 1-4 amino acids or a bond; and

Y is a peptide of 1-4 amino acids or a bond.

22. The method of claim 21, wherein said caspase inhibitor is 2-(Z-amino)benzoyl-Asp-fmk, 2-(Z-amino)-3-methylbenzoyl-Asp-fmk, 2-(Z-amino)-3,5-dimethylbenzoyl-Asp-fmk, 2-(Z-amino)-4-chlorobenzoyl-Asp-fmk, 2-(Z-amino)-5-chlorobenzoyl-Asp-fmk, 2-(Z-amino)-5-fluorobenzoyl-Asp-fmk, 2-(Z-amino)-6-fluorobenzoyl-Asp-fmk, cis-2-(Z-amino)cyclohexanecarboxyl-Asp-fmk, 2-(Z-amino)-5-methylbenzoyl-Asp-fmk, 2-(Z-amino)-6-methylbenzoyl-Asp-fmk, 2-(Z-amino)-6-chlorobenzoyl-Asp-fmk, 2-(Z-amino)-3-methoxybenzoyl-Asp-fmk, 2-(Z-amino)thiophene-2-carboxyl-Asp-fmk, 2-(methoxycarbonylamino)thiophene-2-carboxyl -Asp-fmk, cis-2-(Z-amino)cyclopentanecarboxyl-Asp-fmk, trans-2-(Z-amino)cyclopentanecarboxyl-Asp-fmk, 2-(Z-amino)benzoyl-Asp-DCB-methylketone, methoxycarbonyl-Val-(2-aminobenzoyl)-Asp-fmk, Z-Glu-(2-aminobenzoyl)-Asp-fmk or Z-Val-(2-aminobenzoyl)-Asp-fmk.

23. The method of claim 1, wherein said caspase inhibitor has the formula VI:



(VI)

or a pharmaceutically acceptable salt thereof, wherein

R_1 is an optionally substituted alkyl or hydrogen;

R_2 is hydrogen or optionally substituted alkyl;

R_3 and R_4 independently are hydrogen, optionally substituted aryl, optionally substituted heterocyclic, optionally substituted carbocyclic, optionally substituted heteroaryl, optionally substituted alkyl, optionally substituted alkenyl, or optionally substituted alkynyl;

R_5 is an optionally substituted alkyl, optionally substituted carbocyclic, optionally substituted heterocyclic, optionally substituted aryl or optionally substituted heteroaryl;

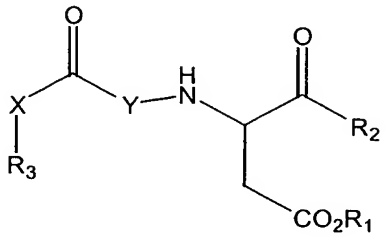
Z is O, S, NR_8, or $(CR_9R_{10})_n$, where R_8, R_9 and R_{10} independently are hydrogen, alkyl or cycloalkyl, and n is 0, 1, 2, or 3; and

X is a peptide of 1-2 amino acids or a bond.

24. The method of claim 23, wherein said caspase inhibitor is 1-(Carbonyl-Asp-CH_2F)ethyl N-phenylcarbamate, 1-(Carbonyl-Asp-CH_2F)ethyl N-benzylcarbamate, 2-Methyl-1-(carbonyl-Asp-CH_2F)propyl N-phenylcarbamate, 2-Methyl-1-(carbonyl-Asp-CH_2F)propyl N-benzylcarbamate, 2-Methyl-1-(carbonyl-Asp-CH_2F)propyl N-(2,6-dichlorophenyl)carbamate, 2-Methyl-1-(carbonyl-Asp-CH_2F)propyl N-(2,5-dichlorophenyl)-carbamate, 2-Methyl-1-(carbonyl-Asp-CH_2F)propyl N-(2,4-dichlorophenyl)-carbamate, 2-Methyl-1-(carbonyl-Asp-CH_2DCB)propyl N-phenylcarbamate, 2-Methyl-1-(carbonyl-Asp-CH_2DCB)propyl N-(2,6-dichlorophenyl)-carbamate, 2-Methyl-1-(carbonyl-Asp-CH_2PTP)propyl *N*-phenylcarbamate, 2-Methyl-1-(carbonyl-Asp-CH_2PTP)propyl *N*-(2,6-dichlorophenyl)-carbamate, 2-Methyl-1-(carbonyl-Asp-CH_2DPP)propyl *N*-phenylcarbamate, 2-Methyl-1-(carbonyl-Asp-CH_2DPP)propyl*N*-(2,6-

dichlorophenyl)-carbamate, 2-Methyl-1-(carbonyl-Asp-CH_2F)propyl *N*-(2-methyl-1-methoxycarbonyl-propyl)carbamate, 2-Methyl-1-(carbonyl-Asp-CH_2F)propyl N-(3-fluorophenyl)carbamate, 2-Methyl-1-(carbonyl-Asp-CH_2F)propyl N-(4-fluorophenyl)carbamate, 2-Methyl-1-(carbonyl-Asp-CH_2F)propyl N-(3,4-difluorophenyl)carbamate, 2-Methyl-1-(carbonyl-Asp-CH_2F)propyl N-(4-phenoxyphenyl)carbamate, 1-(Carbonyl-Asp-CH_2F)propyl N-phenylcarbamate, 1-(Carbonyl-Asp-CH_2F)butyl N-phenylcarbamate, 1-(Carbonyl-Asp-CH_2F)-2-propenyl N-phenylcarbamate, 2-(4-Imidazolyl)-1-(carbonyl-Asp-CH_2F)ethyl N-phenylcarbamate, 2-Phenyl-1-(carbonyl-Asp-CH_2F)ethyl N-phenylcarbamate, 2-Methyl-1-(carbonyl-Asp-CH_2F)butyl N-phenylcarbamate, 3-Methyl-1-(carbonyl-Asp-CH_2F)butyl N-phenylcarbamate, 1-Phenyl-1-(carbonyl-Asp-CH_2F)methyl N-phenylcarbamate, 1-(2-Chlorophenyl)-1-(carbonyl-Asp-CH_2F)methyl N-phenylcarbamate, 1-(4-Chlorophenyl)-1-(carbonyl-Asp-CH_2F)methyl N-phenylcarbamate, 1-Cyclohexyl-1-(carbonyl-Asp-CH_2F)methyl N-phenylcarbamate, 2-Chloro-1-(carbonyl-Asp-CH_2F)ethyl N-phenylcarbamate, 2,2,2-Trifluoro-1-(carbonyl-Asp-CH_2F)ethyl N-phenylcarbamate or Z-Valine 2-methyl-1-(carbonyl-Asp-CH_2F)propyl ester.

25. The method of claim 1, wherein said caspase inhibitor has the formula VII:



(VII)

or a pharmaceutically acceptable salt thereof;

wherein R_1 is an optionally substituted alkyl or hydrogen;

R_2 is hydrogen or optionally substituted alkyl;

R_3 is an alkyl, saturated carbocyclic, partially saturated carbocyclic, aryl, saturated heterocyclic, partially saturated heterocyclic or heteroaryl group, wherein said group is optionally substituted;

X is O, S, NR_4, or $(CR_4R_5)_n$, where R_4 and R_5 are, at each occurrence, independently selected from the group consisting of hydrogen, alkyl and cycloalkyl, and n is 0, 1, 2, or 3; or

X is NR_4, and R_3 and R_4 are taken together with the nitrogen atom to which they are attached to form a saturated heterocyclic, partially saturated heterocyclic or heteroaryl group, wherein said group is optionally substituted; or

X is CR_4R_5, and R_3 and R_4 are taken together with the carbon atom to which they are attached to form a saturated carbocyclic, partially saturated carbocyclic, aryl, saturated heterocyclic, partially saturated heterocyclic or oxygen-containing heteroaryl group, wherein said group is optionally substituted; and

Y is a residue of a natural or non-natural amino acid;

provided that when X is O, then R_3 is not unsubstituted benzyl or *t*-butyl; and when X is CH_2, then R_3 is not hydrogen.

26. The method of claim 25, wherein said caspase inhibitor is 2-Chlorobenzyloxycarbonyl-Val-Asp-fmk, 3-Chlorobenzyloxycarbonyl-Val-Asp-fmk, 4-Chlorobenzyloxycarbonyl-Val-Asp-fmk, Phenethoxycarbonyl-Val-Asp-fmk, Cyclohexylmethoxycarbonyl-Val-Asp-fmk, Methoxycarbonyl-Val-Asp-fmk, Ethoxycarbonyl-Val-Asp-fmk, Isopropyloxycarbonyl-Val-Asp-fmk, 2-Chlorobenzyloxycarbonyl-Ile-Asp-fmk, 3-Chlorobenzyloxycarbonyl-Ile-Asp-fmk, 4-Chlorobenzyloxycarbonyl-Ile-Asp-fmk, Phenylacetyl-Val-Asp-fmk, 4-Nitrobenzyloxycarbonyl-Val-Asp-fmk, 2,5-Dimethylbenzyloxycarbonyl-Val-Asp-fmk, 3,4-Dichlorobenzyloxycarbonyl-Val-Asp-fmk, 3,5-Dichlorobenzyloxycarbonyl-Val-Asp-fmk, 2,5-Dichlorobenzyloxycarbonyl-Val-Asp-fmk, 2,6-Dichlorobenzyloxycarbonyl-

Val-Asp-fmk, 2,4-Dichlorobenzyloxycarbonyl-Val-Asp-fmk, 2,4-Dimethylbenzyloxycarbonyl-Val-Asp-fmk, 4-Ethylbenzyloxycarbonyl-Val-Asp-fmk, 4-Bromobenzyloxycarbonyl-Val-Asp-fmk, 4-Fluorobenzyloxycarbonyl-Val-Asp-fmk, Cyclopentylmethoxycarbonyl-Val-Asp-fmk, 4-Trifluoromethylbenzyloxycarbonyl-Val-Asp-fmk, 3-Phenylpropionyl-Val-Asp-fmk, Benzylaminocarbonyl-Val-Asp-fmk, 3-Phenylpropyloxycarbonyl-Val-Asp-fmk, 2,4-Difluorobenzyloxycarbonyl-Val-Asp-fmk, 3,4-Difluorobenzyloxycarbonyl-Val-Asp-fmk, 4-Morpholinecarbonyl-Val-Asp-fmk, 4-Pyridylmethoxycarbonyl-Val-Asp-fmk, 2-Pyridylmethoxycarbonyl-Val-Asp-fmk, 2,6-Dichlorobenzyloxycarbonyl-Val-Asp-DCB-methylketone, Isobutoxycarbonyl-Val-Asp-fmk, Propionyl-Val-Asp-fmk, Benzyl-glutaryl-Val-Asp-fmk,Glutaryl-Val-Asp-fmk, 3-(2-Phenyloxyphenyl)propionyl-Val-Asp-fmk, 3-(5-Bromo-2-hydroxyphenyl)propionyl-Val-Asp-fmk, 3-Fluorobenzyloxycarbonyl-Val-Asp-fmk, 2-Fluorobenzyloxycarbonyl-Val-Asp-fmk, 3-Methylbenzyloxycarbonyl-Val-Asp-fmk, 2-Chloro-4-fluorobenzyloxycarbonyl-Val-Asp-fmk, 2-Naphthylmethoxycarbonyl-Val-Asp-fmk, *p*-Toluenesulfonyl-Val-Asp-fmk or *p*-Toluenesulfonyl-Phe-Asp-fmk.